Long-Term Renal Function and Cardiovascular Disease Risk in Obese Kidney Donors

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Background and objectives: Increasing demand for live-donor kidneys has encouraged the use of obese donors despite the absence of long-term outcome data and evidence that obesity can adversely affect renal function. We wished to determine whether obesity increased the risk for renal dysfunction and other medical comorbidities in donors several years after donation.

Design, setting, participants, & measurements: Ninety-eight patients who donated a kidney 5 to 40 years previously were stratified according to body mass index (BMI) at donation and evaluated for renal dysfunction and risk factors for cardiovascular disease. Patients who were from the 2005 through 2006 National Health and Nutrition Examination Survey database; did not have renal disease; and were matched for age, gender, race, and BMI served as two-kidney control subjects.

Results: Renal function in obese (BMI ≥30) and nonobese (BMI <30) donors was similar, and both donor groups had reduced renal function compared with BMI-matched two-kidney control subjects. Obesity was associated with more hypertension and dyslipidemias in both donors and two-kidney control subjects; however, there were no significant differences between the two groups within each BMI category.

Conclusions: These results indicate that obese donors are not at higher risk for long-term reduced renal function compared with nonobese donors and that the increased incidence of hypertension and other cardiovascular disease risk factors in obese donors is due to their obesity and is not further exacerbated by nephrectomy. These findings support the current practice of using otherwise healthy overweight and obese donors but emphasize the need for more intensive preoperative education and postoperative health care maintenance in this donor group.

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Live-donor kidney transplantation is generally considered the best choice for patients who have renal failure and are awaiting transplantation, because these kidneys function better than kidneys from deceased donors, and waiting times for deceased-donor transplants are long (1). Although several studies have shown that kidney donation has low short-term morbidity and mortality, the data on long-term outcomes are much less complete (2,3). This is particularly true of donor groups with medical comorbidities that are now being used with greater frequency. One such patient group is the obese donor. There is increasing evidence that obese patients are more susceptible to the development of renal disease either as a direct result of their weight or as a consequence of their obesity-related comorbidities, such as hypertension and diabetes. Conversely, even mild renal dysfunction is thought to increase the risk for developing cardiovascular disease (CVD), an important concern in overweight patients who are already prone to developing this complication (4–6). Despite these findings in the general population, little is known about the effects of obesity on long-term outcomes in kidney donors. This study addressed this issue by examining long-term outcomes in nonobese (body mass index [BMI] <30) and obese (BMI ≥30) patients who donated a kidney at the University of California, San Francisco (UCSF), between 1967 and 2003.

Materials and Methods

Patients

Between 1967 and 2003, 601 individuals who lived in northern California donated a kidney at UCSF as part of the live-donor kidney transplant program. Of these, 207 were successfully located, and 107 agreed to participate in the study. The main reasons for not participating were employment-related time constraints and living far away from the study center. Of those who agreed to participate, nine did not have predonation laboratory values available and were excluded, leaving a study population of 98 donors. All participants provided written informed consent before participation, and the institutional review board at UCSF approved the study.

Two-kidney control subjects were obtained from the 2005 through 2006 National Health and Nutrition Examination Survey (NHANES) database and matched with donors for current BMI, current age, race, gender, diabetes, and smoking history (7). Individuals with known renal disease and other significant medical comorbidities, with the

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exception of hypertension and dyslipidemias, were excluded. Three control subjects were selected for each kidney donor.

Clinical and Laboratory Assessments

All participants were evaluated at the study center by the same study personnel. The study consisted of a medical history; laboratory tests; and physical examination, including height, weight, and BP. BP was measured with a standard mercury sphygmomanometer correctly sized for the participant's upper arm. Measurements were made after 5 min of rest in a sitting position, and the average of two to three measurements was recorded. Hypertension was defined as a systolic BP (SBP) >140 mmHg, a diastolic BP (DBP) >90 mmHg, or a known diagnosis of hypertension (8). BMI was calculated as weight in kg/height in m^2. Patients were divided into two groups according to their BMI at donation using the National Heart, Lung, and Blood Institute guidelines. The nonobese group consisted of normal weight (BMI 18.5 to 24.9) and overweight (BMI 25.0 to 29.9) patients, and the obese group consisted of patients with BMI ≥30 (9,10).

Blood tests included electrolytes, creatinine, total cholesterol, and HDL cholesterol. Twenty-four-hour urine samples were collected for urinalysis, protein, and albumin excretion. Data from the time of donation was obtained from medical records. The Modification of Diet in Renal Disease (MDRD) formula was used to estimate the GFR as described previously (11,12). GFR <60 ml/min per 1.73 m^2 was considered abnormal. Twenty-four-hour total urine protein and albumin excretion were considered abnormal when values were ≥150 and 30 mg/d, respectively (13,14). Serum total cholesterol was considered abnormal when values were >200 mg/dl. Serum HDL levels were considered abnormal when values were <40 mg/dl in men and <50 mg/dl in women (15).

Data for the two-kidney control subjects were collected from the NHANES database and included age, gender, race, height, weight, BP, creatinine, and total and HDL cholesterol (7). The Framingham Risk Score (FRS) was calculated as described elsewhere (16).

Statistical Analysis

Data were analyzed using STATA (Stata Corp., College Station, TX) software. Demographic data were compared among kidney donor BMI groups using the χ^2 test for categorical variables and the paired and unpaired *t* test for continuous variables when appropriate. Normality of distribution for continuous variables was examined, and outliers were inspected and cross-checked against source documents. A scatter plot was used to identify visually relationships between BMI and clinical parameters such as calculated renal function and CVD risk factors at study. Linear regression was used to demonstrate correlation between the variables and was depicted with a line of best fit. The significance of correlation was analyzed using Pearson correlation coefficient. Multivariate logistic regression and linear regression models were created to determine the independent association between BMI at donation and targeted clinical outcomes (renal insufficiency, hypertension, proteinuria, dyslipidemias) while controlling for the covariates. Statistical significance was considered to be $P < 0.05$.

Results

Demographics and Clinical Characteristics

Demographic characteristics of the clinical center population and the study group selected from this population were comparable, although there were a higher percentage of overweight donors (BMI 25.0 to 29.9) in the clinical center population. The percentage of patients with BMI ≥35 at the time of donation was very low in both the study and center populations (Table 1).

The clinical characteristics, including comorbidities such as diabetes and tobacco use, were similar between the obese and nonobese BMI groups (Table 2). Patients were considered to have diabetes when they had previously received a diagnosis of the disease and were taking insulin or oral hypoglycemic agents. None of the patients with diabetes at study had diabetes at the time of donation, and none of the patients had hypertension at the time of donation, because, until recently, this was a contraindication to donation at our institution. BMI tended to increase with time, so 16 (19.5%) of 82 donors who were not obese at the time of donation became obese at follow-up (F/U). All donors who were obese at the time of donation remained obese. The median BMI for the obese donor group at donation was 32.4, and the range was 30.2 to 35.5, with only one patient

Table 1. Clinical center and study population demographics[a]

Demographic	Clinical Center Population	Study Population
No. of patients	601	98
Age at donation/study (yr; mean ± SD)	40 ± 11	44 ± 11
Time since donation (yr; mean ± SD)	12 ± 4	11 ± 7
Female (%)	60	64
Race (%)		
white	56	70
black	8	4
other	36	26
BMI at donation		
<25.0	50	57
25.0 to 29.9	35	27
30.0 to 34.9	13	15
≥35.0	2	1

[a]BMI, body mass index.

Table 2. Current clinical characteristics of donors according to BMI at donation[a]

Parameter	Entire Population	Obese at Donation	Nonobese at Donation		P^b
			Nonobese at F/U	Obese at F/U	
n	98	16	82		NA[c]
			66	16	NA[d]
Age (yr; mean ± SD)	56 ± 10	56 ± 8	56 ± 10		0.893[c]
			56 ± 10	54 ± 9	0.436[d]
Age at donation (yr; mean ± SD)	44 ± 11	45 ± 9	44 ± 11		0.736[c]
			46 ± 11	38 ± 9	0.0113[d]
Female (%)	64	50	67		0.2[c]
			67	69	0.873[d]
Follow-up (yr; mean ± SD)	11 ± 7	10 ± 3	11 ± 7		0.439[c]
			10 ± 6	15 ± 10	0.0063[d]
BMI at donation (mean ± SD)	25 ± 4	32 ± 2	24 ± 3		0.0001[c]
			23 ± 2	27 ± 3	0.0001[d]
BMI at study (mean ± SD)	28 ± 5	36 ± 3	26 ± 4		0.0001[c]
			25 ± 2	33 ± 2	0.0001[d]
Donors with diabetes (*n*)	3	1	2		NA[c]
			1	NA	NA[d]
Current or previous smoker (*n*)	4	2	2		NA[c]
			1	NA	NA[d]

[a]Nonobese = BMI <30, obese = BMI ≥30. F/U, follow-up.
[b]Measured by *t* test for continuous variables and the χ^2 test for categorical variables.
[c]Comparisons between obese at donation and nonobese at donation groups.
[d]Comparisons between nonobese at F/U and obese at F/U.

having a BMI ≥35. On F/U, the median BMI in the obese group had increased to 36.2, the range was 30.1 to 40.4, and 10 patients had BMI ≥35. These 10 patients underwent additional subset analysis to determine whether they were at higher risk for developing complications.

Renal Function

Donor renal function before donation and at study is depicted in Table 3. As expected, all patients had predonation GFR >60 ml/min per 1.73 m^2 (range 60.2 to 154.1 ml/min per 1.73 m^2), and there were no significant differences in mean predonation values between the two BMI groups or between nonobese donors who became obese at F/U. Although current mean GFRs were significantly reduced ($P = 0.0001$), there were no differences between any of BMI groups. Overall, 43% of donors developed reduced renal function (GFR <60 ml/min per 1.73 m^2). In multivariate logistic regression adjusted for gender, F/U years, age, and predonation GFR, obesity at time of donation was not a significant risk factor for reduced renal function (odds ratio [OR] for obese donors 2.6; $P = 0.161$). Age and lower GFR at donation remained independent risk factors for developing reduced renal function (OR 1.16 [$P = 0.0001$] and OR 0.96 [$P = 0.024$], respectively). When GFR was compared between donors and their corresponding BMI-matched two-kidney control subjects, both obese and nonobese donor groups had significantly reduced GFRs compared with their two-kidney control groups. In contrast, the differences in GFR between BMI groups within each population were small (Table 4, Figure 1).

Proteinuria

None of the study patients had abnormal urinary protein excretion at donation (Table 3). At study, the mean urinary protein excretion had increased to 119 ± 55 mg/d in the entire cohort, and 22% had abnormal proteinuria. Significantly more obese donors had abnormal proteinuria when compared with nonobese donors (44 *versus* 18%; $P = 0.03$). In contrast, nonobese donors who became obese at F/U did not have significantly more abnormal proteinuria compared with nonobese donors who remained nonobese at F/U.

In multivariate analysis adjusting for BMI, age, F/U, gender, and predonation urinary protein excretion, obesity at the time of donation and male gender were the only independent predictors of abnormal proteinuria (OR 8.9 [95% confidence interval (CI) 1.1 to 70.0; $P = 0.039$] and OR 15.0 [95% CI 2.1 to 104.0; $P = 0.006$], respectively).

Table 3. Current renal function in donors according to BMI at donation[a]

Parameter	Entire Population	Obese at Donation	Nonobese at Donation		P^b
			Nonobese at F/U	Obese at F/U	
Predonation GFR (ml/min per 1.73 m^2; mean ± SD)	87 ± 18	87 ± 14	87 ± 19		0.972[c]
			86 ± 19	95 ± 18	0.153[d]
Current GFR (ml/min per 1.73 m^2; mean ± SD)	63 ± 13	64 ± 13	63 ± 13		0.826[c]
			63 ± 12	66 ± 18	0.353[d]
% with current GFR <60	43	50	41		0.53[c]
			42	38	0.719[d]
Predonation 24-h urine protein excretion (mg/d; mean ± SD)	70 ± 43	80 ± 30	68 ± 45		0.407[c]
			66 ± 44	78 ± 53	0.557[d]
Current 24-h urine protein excretion (mg/d; mean ± SD)	119 ± 55	146 ± 62	113 ± 52		0.027[c]
			112 ± 49	116 ± 66	0.814[d]
% with current 24-h urine protein excretion ≥150 mg/d	22	44	18		0.03[c]
			18	13	0.628[d]
Current 24-h urine albumin excretion (mg/d; mean ± SD)	10 ± 12	17 ± 22	8 ± 8		0.0056[c]
			8± 8	7 ± 7	0.84[d]
% with current 24-h urine albumin excretion ≥30 mg/d	7	13	6		0.428[c]
			6	8	0.84[d]
Predonation serum Cr (mg/dl; mean ± SD)	0.86 ± 0.19	0.88 ± 0.19	0.86 ± 0.19		0.737[c]
			0.87 ± 0.19	0.77 ± 0.13	0.104[d]
Current serum Cr (mg/dl; mean ± SD)	1.11 ± 0.23	1.15 ± 0.21	1.10 ± 0.23		0.458[c]
			1.10 ± 0.22	1.10 ± 0.29	0.85[d]

[a]Nonobese = BMI <30; obese = BMI ≥30. GFR calculated using the Modification of Diet in Renal Disease formula. Cr, creatinine.
[b]Measured by *t* test for continuous variables and the χ^2 test for categorical variables.
[c]Comparisons between obese at donation and nonobese at donation groups.
[d]Comparisons between nonobese at F/U and obese at F/U.

Albuminuria

Predonation albumin excretion values were available only for nine patients and were normal, ranging from 3 to 14 mg/d. Absolute albuminuria was significantly increased in obese donors. The percentage of donors with abnormal albuminuria (albumin excretion >30 mg/d) was also increased in the obese group, but this did not reach statistical significance (Table 3). Obesity was not a risk factor for developing pathologic albuminuria (OR 1.7; 95% CI 0.3 to 10.0; $P = 0.574$); however, in multivariate linear regression, obesity was associated with an 8.7-mg/d increase in total albumin excretion (coefficient 8.7; 95% CI 2.0 to 15.0; $P = 0.01$).

CVD Risk Factors

Hypertension. None of the donors had hypertension at the time of donation. At study, obese donors had significantly higher mean SBP and DBP than nonobese donors, and there were significantly more donors with hypertension in the obese group. Significantly increased SBP and DBP were also seen in nonobese donors who became obese on F/U compared with donors who remained nonobese of F/U, but the differences in hypertension did not reach significance (Table 4). After adjustment for age at donation, gender, family history of hypertension, smoking, and time since donation, obesity at donation remained a significant independent risk factor for developing hypertension over time (OR 4.02; 95% CI 1.20 to 13.00; $P = 0.021$). When BP was compared between donors and two-kidney control subjects, donors in both BMI groups had significantly higher mean DBP than the corresponding two-kidney control subjects, but there were no differences between the mean SBP or incidence of hypertension in either BMI group. (Table 5).

Dyslipidemias. The mean total cholesterol was not significantly different between any of the BMI groups; however, obese donors had significantly lower mean HDL cholesterol levels compared with the nonobese group, and more obese donors had abnormally low HDL levels. HDL levels in nonobese donors who became obese on F/U were lower than in donors who remained nonobese, but the percentages with ab-

Table 4. Current CVD risk factors in donors according to BMI at donation[a]

Parameter	Entire Population	Obese at Donation	Nonobese at Donation		P[b]
			Nonobese at F/U	Obese at F/U	
SBP (mmHg; mean ± SD)	129 ± 16	137 ± 18	127 ± 15		0.0278[c]
			125 ± 14	135 ± 18	0.0179[d]
DBP (mmHg; mean ± SD)	82 ± 10	86 ± 9	81 ± 9		0.0451[c]
			79 ± 9	87 ± 8	0.0032[d]
% with hypertension[e]	40	69	34		0.01[c]
			32	44	0.373[d]
Total cholesterol (mg/dl; mean ± SD)	203 ± 33	206 ± 26	203 ± 34		0.689[c]
			201 ± 35	207 ± 31	0.522[d]
HDL cholesterol (mg/dl; mean ± SD)	59 ± 17	51 ± 13	60 ± 18		0.04[c]
			62 ± 18	54 ± 15	0.0911[d]
% with abnormal HDL cholesterol[f]	19	44	15		0.013[c]
			14	19	0.613[d]
10-yr FRS	5.1	6.3	5.0		0.395[c]
			4.9	5.7	0.58[d]

[a]Nonobese = BMI <30; obese = BMI ≥30.
[b]Measured by t test for continuous variables and the χ^2 test for categorical variables.
[c]Comparisons between obese at donation and nonobese at donation groups.
[d]Comparisons between nonobese at F/U and obese at F/U.
[e]SBP >140 mmHg, DBP >90 mmHg, or known diagnosis of hypertension.
[f]HDL < 40 mg/dl for men, <50 mg/dl for women.

normal HDL levels were similar (Table 4). After adjustment for age, F/U, and smoking, obesity at donation remained a significant risk factor for abnormal HDL levels at study (OR 4.5; 95% CI 1.3 to 15.0; $P = 0.015$). In contrast, when donors were compared with BMI-matched two-kidney control subjects, there were no significant differences in lipid profiles between the donor and control groups within each BMI category (Table 5, Figure 1).

Framingham Risk Score. The FRS was developed using data from the Framingham Heart Study to estimate the 10-yr risk for developing significant CVD outcomes such as myocardial infarction and coronary death (16). The components of the score include age, gender, HDL cholesterol, total cholesterol, SBP, and smoking status. When the FRS was applied to our study groups, donors who were obese at donation had a higher score than nonobese donors, but this difference was NS (Table 4). Similarly, no significant differences were observed between donor and two-kidney control subjects within each BMI group (Table 5, Figure 1).

Subset Analysis of Donors with BMI ≥35 on F/U

A subset of donors ($n = 10$) who had the highest BMI at donation (range 31.3 to 35.5) and whose BMI had increased to ≥35 at F/U (range 35.1 to 40.4) were analyzed separately to determine whether they were at higher risk for long-term complications. This subgroup had a similar current mean GFR and incidence of GFR <60 ml/min per 1.73 m^2 as the nonobese group (62.3 *versus* 63.0 [$P = 0.81$] and 50 *versus* 41% [$P = 0.61$], respectively). The incidence of proteinuria ≥150 mg/d and albuminuria ≥30 mg/d was increased compared with the nonobese group, but the differences were NS (40 *versus* 18% [$P = 0.12$]; and 20 *versus* 6% [$P = 0.19$], respectively). The incidence of hypertension was increased in these donors as compared with nonobese donors (80 *versus* 34%; $P = 0.005$) but was similar to the incidence of hypertension in BMI-matched two-kidney control subjects (80 *versus* 60%; $P = 0.236$). The incidence of abnormally low HDL levels in donors with current BMI ≥35 was similar to that of BMI-matched two-kidney control subjects (40 *versus* 43%; $P = 0.853$) and was higher than in nonobese donors, although this was not statistically significant (40 *versus* 15%; $P = 0.07$).

Discussion

During the past decade, the proportion of obese kidney donors has increased dramatically in most transplant centers in the United States (17). This change reflects the increasing prevalence of obesity in the United States (9), as well as the higher demand for live-donor kidneys with resultant pressure to use obese donors and other donor groups with compensated medical problems, such as mild hypertension (18). Although short-term outcomes with obese donors seem to be comparable to those with nonobese donors, the long-term (>1 yr) consequences of kidney donation by obese donors are not known

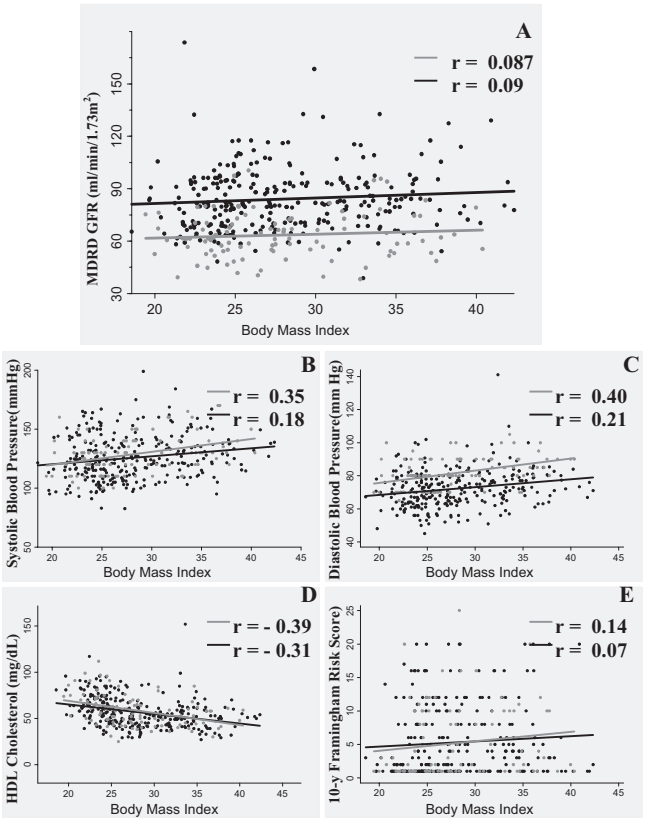

Figure 1. Linear regression analysis of renal function and cardiovascular disease risk factors in donors and matched two-kidney control subjects. (A) Renal function (ml/min per 1.73 m^2) as calculated using the Modification of Diet in Renal Disease (MDRD) equation. (B) Systolic BP (mmHg). (C) Diastolic BP (mmHg). (D) Serum HDL level (mg/dl). (E) Ten-year Framingham Risk Score for significant cardiovascular events. Individual donor (grey circles) and two-kidney control subject (black circles) values as well as the corresponding linear regression lines (gray line: donors; black line: two-kidney control subjects) are depicted. Pearson correlation coefficient (*r*) is shown for each group.

(19–22). This is particularly important in light of the emerging evidence indicating that obesity is a risk factor for development of renal insufficiency, both directly and as a consequence of several obesity-related comorbidities such as hypertension and diabetes (23–25).

In this study, we examined the effects of obesity at donation on clinical outcomes in patients who underwent donor nephrectomy at UCSF between 5 and 40 yr ago. When renal function was measured, all donors had significantly reduced calculated GFRs compared with matched two-kidney control subjects; however, there were no significant differences in GFRs between obese and nonobese donor groups, and the percentage of donors with calculated GFRs <60 ml/min per 1.73 m^2 was also comparable between the donor groups. When proteinuria and albuminuria were evaluated, there was a trend toward higher levels of total urine protein and albumin in donors who were obese at donation, and the incidence of abnormal proteinuria was significantly increased in the obese; however, the incidence of abnormal albuminuria was not significantly different between the two groups. These findings corroborate other reports demonstrating relative preservation of renal function in obese donors on short-term follow-up (19,20,22) but differ from several large-scale population studies that examined the impact of obesity on renal function. For example, a recent study which examined >350,000 adult patients in the Kaiser-Permanente system in California found that obese patients had a significantly increased incidence of ESRD compared with normal-weight control subjects. This association persisted even when corrections were made for variables such as age, gender, BP, diabetes, tobacco use, and dyslipidemias (6). Similarly, a study that examined the consequences of unilateral nephrectomy found that obese (BMI >30) patients who had normal renal function and underwent unilateral nephrectomy for reasons other than donation had much higher rates of renal dysfunction after surgery than patients with BMI <30 (92 *versus* 12%, respectively). The elapsed time between nephrectomy and development of these abnormalities was comparable to the F/U period in our study (10 ± 6 and 9 ± 8 yr, respectively) (26). Finally, it has been reported that renal dysfunction, particularly obesity-related glomerulopathy, improves with weight loss (27). Several possible explanations exist for our differing results. First, it is generally accepted that kidney donors are healthier than the average population, and the effects of obesity on renal function in this population may thus be less pronounced than in the overall population (28–30). Second, renal function was measured with the MDRD formula, which is not as accurate as direct test of GFR and may not detect small differences in renal function (12,31). Third, our sample size was small compared with the center population, increasing the possibility of selection bias. This last concern is addressed in part by comparing obese and nonobese donors within the same cohort; nonetheless, the possible influence of this confounding factor needs to be taken into consideration when examining our results.

Both obesity and moderate renal dysfunction increase the risk for CVD (23,24,32–35). Whether this effect is also present in obese but otherwise healthy individuals who have donated a kidney is not known but is possible because the degree of renal dysfunction that is associated with increased CVD risk (GFR <60) is frequently seen after donation (21,28,29,36–38). Hypertension and dyslipidemias are two important CVD risk factors that correlate with both renal dysfunction and obesity (15,24,39–42). In this study, we found that obese donors were more likely to have hypertension and abnormal HDL levels after donation than nonobese donors; however, when donors were compared with BMI-matched two-kidney control subjects to determine whether this increase was due to nephrectomy, obesity, or a combination of both, the rates of hypertension and lipid abnormalities in obese donors were similar to the rates observed in the obese two-kidney control subjects, suggesting that the increased risks were attributable to obesity rather that nephrectomy. Similarly, when the FRS was used to estimate the 10-yr risk for CVD, no significant differences were found between donors and BMI-matched two-kidney control subjects. It is interesting that patients who were already obese at donation

Table 5. Current clinical parameters in donors and matched two-kidney control subjects[a]

Parameter	Donor BMI Category[b]	Donors	Two-Kidney Control Subjects	P[c]
Renal function				
GFR (ml/min per 1.73 m^2; mean ± SD)	Nonobese	63 ± 13	84 ± 18	0.0001
	Obese	64 ± 13	87 ± 18	0.0001
% with GFR <60	Nonobese	41.0	5.5	0.0001
	Obese	50.0	2.1	0.0001
BP				
SBP (mmHg; mean ± SD)	Nonobese	127 ± 15	124 ± 20	0.251
	Obese	137 ± 18	133 ± 13	0.454
DBP (mmHg; mean ± SD)	Nonobese	81 ± 9	71 ± 11	0.0001
	Obese	86 ± 9	77 ± 10	0.003
% with hypertension[d]	Nonobese	34	38	0.486
	Obese	69	62	0.61
Other CVD risk factors				
Total cholesterol (mg/dl; mean ± SD)	Nonobese	203 ± 34	207 ± 36	0.351
	Obese	206 ± 26	212 ± 38	0.623
HDL cholesterol (mg/dl; mean ± SD)	Nonobese	60 ± 18	58 ± 17	0.391
	Obese	51 ± 13	50 ± 14	0.835
% with abnormal HDL cholesterol[e]	Nonobese	15	24	0.066
	Obese	44	36	0.592
10-yr FRS	Nonobese	5.0	5.1	0.893
	Obese	6.3	6.5	0.881

[a]Nonobese = BMI <30; obese = BMI ≥30. CVD, cardiovascular disease; DBP, diastolic BP; FRS, Framingham Risk Score; SBP, systolic BP.
[b]At time of donation
[c]Measured by t test for continuous variables and the χ^2 test for categorical variables.
[d]SBP >140 mmHg, DBP >90 mmHg, or known diagnosis of hypertension.
[e]HDL <40 mg/dl for men, <50 mg/dl for women.

had more abnormalities in BP, lipid levels, and proteinuria than donors who were nonobese at donation but became obese on F/U, even though the latter group had a greater increase in BMI between donation and F/U. This suggests that the duration of obesity after nephrectomy plays a role in the development of these comorbidities. Alternatively, it is possible that obesity during the acute decrease in renal function associated with nephrectomy may have a greater effect on these measures than obesity that develops once postdonation renal function has stabilized.

One survey reported that many transplant centers set a BMI cutoff of 35 for donor eligibility (43). At our center, the number of donors who had BMI ≥35 and were >5 yr from donation was very low, limiting our ability to analyze this group. This is probably because the prevalence of obesity in the United States has increased most dramatically in the past 5 to 10 yr (44). To provide information about our most obese donors, we examined outcomes in a subset of donors who had the highest BMI at donation and went on to develop BMI ≥35 on F/U. These patients had more proteinuria and hypertension compared with nonobese donors, but the incidence of these abnormalities was not significantly different from matched two-kidney control subjects. Although this subgroup is different from a population of donors who had BMI ≥35 at donation, these results provide additional evidence that the increased incidence of CVD risk factors seen in obese donors is due to obesity rather than nephrectomy.

As with many other analyses of donor outcomes, an important limitation of this study is that our population-based NHANES control group may have had significant health differences from our donor group, which was carefully screened for medical comorbidities at the time of donation. We attempted to correct for these potential differences by excluding control subjects with known renal disease or other significant medical comorbidities, but a control group consisting of patients who were evaluated at the same time as the donors but were excluded for nonmedical issues may provide a more accurate comparison.

Conclusions

Our results demonstrate that donors who are obese at the time of donation do not have a higher incidence of long-term reduced renal function compared with nonobese donors. In addition, donors who are obese at the time of donation have a higher incidence of CVD risk factors, but this increase is attributable to obesity rather than nephrectomy. These findings support the current practice of using otherwise healthy obese donors but emphasize the need for more intensive preoperative

education and postoperative health care maintenance in this donor group.

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Disclosures

None.

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